occupation usually clears up the condition. Phosphorus vapors in former years were a great problem in the manufacture of matches, but since the use of amorphous sulphur has been established, the trade is not so dangerous.

The trade of metal grinding has been greatly improved by the modern invention of a special apparatus for carrying off the dust and by the prohibition of dry grinding. This dust produces an irritation of the mucous membranes of the nose and causes boils. A certain degree of tolerance to the dust may be established, but the nasal processes become atrophied. The feeling of dryness in the throat gives rise to the ingestion of large amounts of alcoholics among the workers. Rheumatic diseases are very common. Cleanliness to a high degree and breathing through the nose are very important in these industries.

The shortening of the working day and pauses in the work so as to enable the men to spend a short time in an atmosphere free from dust have been recommended in all industries where dust and fume are evolved. The ten-hour day for men and the eight-hour day for youths have been strongly advocated.

Almost all cases of industrial disease could be aided if more attention were paid to personal hygiene, both in the home and in the factory, and if the working people would understand the value of out-door exercise there would be very little difference noted in the health of persons working at different occupations, and the expression "occupational diseases" would lose its significance.

ACUTE PULMONARY TUBERCULOSIS

By JOHN B. HUBER, A.M., M.D.

(*Continued from page 30*)

PART II

II. ACUTE PULMONARY MILIARY TUBERCULOSIS

Here the miliary tubercles are in the main confined to the tissues of the lungs and pleura; and the infection is certainly in most cases hematogenous.

Etiology. In children there may have been a history of an acute nfection, such as pertussis or measles; or of some pulmonary or pleural affection. In adults we search for the history of a latent tuberculosis, or of a preëxisting focus, perhaps apical.

The pathology. There may be a tubercular nodule in the course of the intestinal lacteals which are drained by the thoracic duct; or

broken down tubercles in the thoracic duct may discharge into that duct; thence bacilli are discharged with the lymph and chyle into the subclavian veins. Thus, in the venous circulation the tubercle bacilli reach the right ventricle, to be conveyed thence by the pulmonary arteries to the lungs; the bacilli are then filtered out in the terminal capillaries which are enmeshed with the air vesicles, in and about which the miliary tubercles form; a latent focus in the lung (very especially in bronchial or tracheal glands) or in the pleura, has broken down into a pulmonary vessel; and the miliary tubercles result. These are at first grey, pearly translucent and afterward yellow and opaque; they are in size from a millet seed, or a lentil, to a hazel nut. External to the tubercle is a zone of catarrhal inflammation, which is like to extend to neighboring alveoli and bronchioli. The bronchi may contain a frothy mucus. The whole lung is hyperemic and heavier than normal; it may be quite studded with tubercles, and may have the feel of a bag of nuts. The spleen is likely to be enlarged.

The symptoms. The invasion is like that of acute bronchitis, though the gravity of the disease soon becomes manifest. The patient becomes markedly apprehensive. There is chill and fever, malaise and headache. The cough is like to be frequent and distressing; the sputum is at first mucous and perhaps scanty, then mucopurulent and perhaps blood streaked. Hemoptysis is an important sign; seen early in the disease it results from the breaking down of a tubercle involving the walls of a blood vessel. Such hemoptysis may be an initial symptom.

Very marked dyspnoea and cyanosis, out of all proportion to the physical signs, are pathognomonic; they indicate clogged up bronchioli and tuberculous (and therefore non-functioning) alveoli. The respirations may increase to sixty.

The temperature is irregular, like to be hectic and may rise to 103; sweating accompanies temperature falls.

The pulse becomes rapid, feeble, variable, soft, easily compressible; the blood pressure is much reduced.

There is anorexia and impaired digestion with rapid loss of flesh and strength. There is pain, probably due to pleural miliary tubercles. The face is pallid, in odd contrast with the hot skin.

The symptoms in children are very like those of acute bronchopneumonic tuberculosis.

The physical signs are slight by comparison with the gravity of the disease; they are those of a general acute bronchitis with accompanying pleurisy: in children the signs are of bronchitis and bronchopneumonia.

One observes on inspection the pallor and the rapid loss of flesh

and strength. There is bronchial fremitus; and a friction rub, on both inspiration and expiration, due to the pleural involvement. Percussion will hardly evidence the miliary tubercles; dullness at the apices or at the bases would indicate old tubercular foci; a hyperresonant note would indicate patches of emphysema. The respirations are rude or sonorous or bronchial. Fine dry or moist râles may be heard over both sides, especially after coughing. We may palpate the spleen.

The diagnosis is made by the history of glandular or bone tuberculosis, or of pertussis or measles in children; the acute bronchitis with great dyspnoea and cyanosis; tubercle bacilli in the sputum; nodules in the choroid; X-ray shadows of nodules and of enlarged bronchial and tracheal glands.

The prognosis: There is swift and uninterrupted decline, with death in from several days to two months. It is considered that this form may possibly terminate in chronic phthisis.

III. ACUTE PNEUMONIC TUBERCULOSIS

Any of the predispositions tending to tuberculosis in general are causative of acute pneumonic tuberculosis; profound emotional disturbance has been noted in the etiology. The history of pleurisy or of an old tuberculous focus may be elicited.

The pathology. A whole lobe or even a whole lung is solidified; after this softening and caseation are very acute. There is not so often one large cavity as in chronic tuberculosis, as there are small numerous honeycombing cavities. And there is very little connective tissue proliferation about the walls, or other evidences of reparative processes as in chronic tuberculosis. It is probable that "mixed infection" is largely responsible for the rapid course of the disease. One speaks of caseous pneumonia; we find a section of intense dark-red congestion or the granular, greyish yellow appearance indicative of the red and grey hepatization in lobar pneumonia. These appearances of the lesion, however, are due not so much to the formation and breaking down of the exudate (as in lobar pneumonia); but they are the caseation and necrosis processes characteristic of pulmonary tuberculosis. The lesions of lobar pneumonia and of acute pneumonic tuberculosis have this in common: that tubercule bacilli have been demonstrated by Prudden to produce not only tubercles but also exudative pneumonia. An old lesion may be found from which the acute process has started. The pleura is dull in color and covered with a thin exudate.

The symptoms. The case begins as does lobar pneumonia. The invasion is sudden, with rigor, abrupt chill, a stabbing pain in the side, then comes fever, irregular, with morning remissions and evening

exacerbations, of the "inverse" type. Cough is frequent and distressing, with sputum at first scanty and thin, later purulent, greenish or rusty, perhaps blood streaked and perhaps later containing the tubercle bacilli and elastic fibers from necrotic areas.

There may be hemoptysis. The dyspnoea and cyanosis are very marked; one fears suffocation. The pulse begins rapid and full; and becomes rapid and feeble as the patient's strength is dissipated. Sweating is profuse after an abrupt temperature drop. The patient is anxious; he has headache, his digestion is bad, constipation is the rule. The urine is an acute infection urine, scanty, high-colored, of high specific gravity. The loss of flesh and strength is very rapid. Instead of the crisis, which generally precedes recovery in lobar pneumonia, the patient's fever and other symptoms continue, becoming progressively worse; death is the usual ending after several weeks to three months, even in six days, as has been reported.

The *physical signs* are much as in lobar pneumonia; they may be found over one lobe, upper or lower, or over an entire lobe. The signs are of rapid softening in the interior of consolidated areas. The respirations are very frequent. There may be bronchial fremitus; and friction fremitus from pleural involvement. There is dullness. The breathing is bronchial; there is bronchophony. Sub-crepitant râles are marked.

The *diagnosis* is between this disease and lobar pneumonia. Failure here has been frequent, and not unnaturally, since the onset is very similar, and one thinks he has a straight pneumonia to deal with until the time for the crisis passes, with no crisis and no evidence of resolution; even then one thinks the case is but one of delayed resolution. The difficulty is increased by the fact that the tubercle bacilli are rarely found in the sputum the first fortnight of the disease. One should observe the irregular hectic temperature of tubercular pneumonia; the aggravation rather than the subsidence of the symptoms, after eight or nine days (the time of the crisis); and the physical signs of acute softening. The sputum should be examined every day for tubercle bacilli and necrosis products. The history of a previous tuberculosis should be very suggestive of an acute tubercular lesion.

The prognosis. The patient becomes progressively worse and dies in from a week to three months; cases are reported in which the disease has turned into a chronic pneumonia.

IV. ACUTE BRONCHOPNEUMONIC TUBERCULOSIS

Perhaps most of the cases of this form of tuberculosis are in children, and especially coincident with or after an acute exanthem, as measles

or pertussis. The factors predisposing to tuberculosis in general would obtain here.

The pathology. The tubercle bacilli enter the smaller bronchi by inhalation, by aspiration from an existing tubercular lesion, at the apex or elsewhere, from pulmonary lymph nodes, or by way of the blood vessels. The bronchial and tracheal glands and those at the roots of the lungs are (especially in children) enlarged, inflamed and even caseated. The bronchioli are clogged with purulent exudate; the vesicles are filled with the products of catarrhal inflammation. The bronchial walls show coagulation necrosis; there are caseous peribronchial tubercles and there is cheesy exudate into the surrounding parenchyma. There are scattered reddish grey, yellowish white areas of peri-bronchial consolidation, at first separated by normal tissue; the process advances into normal tissue by infiltration in the borders of the caseous areas. Soon the diseased areas become confluent, progressively softening, until a large part of one or both lungs may exhibit a massive lesion. Necrosis, "gelatization," and cavitation will result, this process being hastened by the degree of mixed infection present.

The symptoms. In children at the height of measles or whooping cough the bronchitis becomes greatly emphasized, and the pnenomena of an acute bronchopneumonia develop. In adults the symptoms are those of an ordinary bronchopneumonia; but much more severe. The onset here is less acute than in an acute pneumonic tuberculosis, or in lobar pneumonia. There are premonitory, perhaps repeated chills; there is fever but not so rapid in rise nor so extreme as in acute pneumonic tuberculosis. The pulse is rapid, later feeble; there is dyspnoea, soon becoming very marked, with cyanosis. There is cough, with at first mucus, and then purulent sputum, containing perhaps tubercle bacilli, elastic fibers and other necrosis products. There is generally pain in the chest. Headache will point to a meningeal lesion. The pneumonia becomes grave, with rapid extension of the tubercular process; mixed infections will give hectic symptoms. There is progressive loss of flesh and strength; death within six weeks.

The physical signs are bilateral and indicate bronchitis, with areas of consolidation, at first discrete and later confluent. Dyspnoea and cyanosis are evident. There is bronchial fremitus; dullness over consolidation, softening and cavitation, especially at the apices. The breathing is bronchial, the voice gives bronchophony, there are numerous sub-crepitant râles.

The diagnosis. An ordinary bronchopneumonia may be assumed until persistence of the symptoms and their unusual gravity suggest the tubercular type. A family or previous history of tuberculosis is

suggestive. The tubercle bacilli in the sputum, with the rapid decline, will settle the diagnosis. Typhoid fever has not so extensive a bronchitis, nor pulmonary lesions so marked nor tubercle bacilli in the sputum. In acute bronchopneumonic tuberculosis there are no meteorism, no positive Widal, no rose spots, etc.

The prognosis. The fatal bronchopneumonia cases are very frequently tubercular; death is in from three weeks to three months. Cases of development into chronic tuberculosis have been reported.

The treatment. One of the most melancholy things about medicine is that descriptions of very grave diseases may be very elaborate and detailed until the section on therapy is reached. Then but a few words, and those but inconclusive, are all that can be offered. Acute tuberculosis almost invariably ends fatally; wherefore the therapy must be largely symptomatic. The prophylaxis and treatment must be those of acute affections, such as of pneumonia or bronchopneumonia. The measures appropriate in ordinary tuberculosis will apply as well in acute tuberculosis. A very essential prophylactic measure is the disposition of the sputum, which may contain the tubercle bacillus. Such prophylaxis is just as imperative as in pulmonary tuberculosis. In acute tuberculosis we must rely on hygiene, hydrotherapy, fever dietary drugs, and perhaps blood letting and good nursing. Serum therapy may avail against mixed infections. The emunctories (bowels, kidneys and skin), must be made to functionate as well as possible. We give heart stimulants by the mouth and the hypodermatic needle; expectorants, guaiacol, styracol, eucalyptol, oxygen, alcohol, and anodynes freely in view of the almost hopeless prognosis, when the diagnosis is positively established.

HOW CAN THE PRIVATE DUTY NURSE BEST CONSERVE HER STRENGTH AND INCOME

By ADELAIDE L. SHARPE
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The nurse doing private duty differs from those in other positions, in that there is more necessity for watchfulness in the care of her health and resources. The statement so often made that nurses learn this only from personal experience is not true. The majority would be glad if really practical suggestions were given them, and if they could be properly advised how to avoid mistakes which lead to many of the conditions they see in others. I will admit they often fail to follow the old saw: "You must economize!" as it is a generalization with no definite plan to follow.